

Post polio syndrome (PPS) was diagnosed in 26% of the patients. They had all increasing muscle weakness with new atrophy. Polio-related loss of function including cervical and lumbosacral radiculopathies, mononeuropathies and degenerative joint disease were found in an additional 53%. 11 patients (13%) had distinct non-polio-related disorders that caused new loss of function, including CNS-lesions and depression. The remaining 8% had a stable condition.

Conclusion The majority of polio patients who seek hospital experience a new loss of function due to polio-related disorders. A careful neurological examination is necessary to identify the correct diagnosis and treatment.

SC 130

Effect of spinal cord stimulation on severe spasticity in patients with traumatic spinal cord injury

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Introduction The purpose of this study was to evaluate the effect of spinal cord stimulation (SCS) on severe spasticity of the lower limbs in patients with traumatic spinal cord injury (SCI) under close scrutiny of the site and parameters of stimulation.

Method 10 SCI patients (4 women, 6 men) were included in the study. Levels of spasticity before and during stimulation were compared according to a clinical rating scale and by surface electrode myoelectricity (pEMG) during passive flexion and extension of the knee, supplemented by a pendulum test with the stimulating device switched either on or off over an appropriate period.

Results Both the clinical and the experimental parameters clearly demonstrated that SCS, when correctly handled, is a highly effective approach to controlling spasticity in the spinal cord injury subjects. The success of this type of treatment hinges on four factors: (i) the epidural electrode must be located over the upper lumbar cord segment (L1, L2, L3); (ii) the train frequency of stimulation must be in the range of 50–100 Hz, the amplitude within 2–7 Volts and the stimulus width of 210 µsec; (iii) the stimulus parameters must be optimised by clinically assessing the effect of arbitrary combinations of the four contacts of the quadripolar electrode; (iv) amplitudes of stimulation must be adjusted to different body positions.

Conclusion Severe spasticity affecting the lower extremities of patients with chronic spinal cord injuries can be effectively suppressed via stimulation of the upper lumbar cord segment.

SC 130a

First experience with the "LOKOMAT" gait orthosis in post-acute brain-injured patients

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Patients with severe brain injury may develop seriously disabling movement disorders, which may be due to lesions of the corticospinal pathways as well as extrapyramidal dysfunction. Lengthy immobilisation also affects somatosensory afferents and body image. Therapeutic application of a mechanical gait orthosis is a new approach in the management of impaired motor control and postural instability in neurological patients. We present our first experience using the "LOKOMAT" in a

patient with post-traumatic spastic quadriplegia with left predominance, who was examined prior to, during, and after a three-week training period. The Functional Ambulatory Categories (FAC) improved from 1 to 2. The Ashworth Score improved from 3 to 1 in triceps surae and from 2 to 1 in hamstring muscles on the more affected side. Notably, muscle strength improved on the less affected side in triceps surae (4 to 5), quadriceps (4 to 5), hamstrings (3 to 4) and gluteus maximus (3 to 4, according to Oxford Scale). The 10-meter walking time deteriorated temporarily from 7.09 to 8.12 minutes, but subsequently improved to 5.12 minutes. The same pattern occurred in the 6-minute walking test, with distances of 8.3, 7.3, and 10.5 meters, respectively. Functional evaluation using the Rivermead Visual Gait Assessment (RVGA) revealed similar results: 28, 31, and 16 points, respectively. The transient functional deterioration may be explained by the necessity to replace pathological locomotor patterns and to adapt to a new, more physiological motor programme. The overall improvement, as measured by various scales, underscores the efficacy of the "LOKOMAT" as a new and promising adjunct to neurorehabilitation.

Results of the evaluation of four additional brain-injured patients will be presented.

Child neurology

SC 131

Effect of the immunosuppressant drug FK506 on neonatal cerebral mitochondrial activities and energy metabolism after transient intrauterine ischemia in rats

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Introduction The immunosuppressant drug FK506 reduced neocortical infarct size due to middle cerebral artery occlusion in adult animals. In the immature brain, the effect of this immunosuppressant during ischemia and reperfusion is, however, unclear. In the present study, mitochondrial respiratory activities and energy metabolism were measured in neonatal rat brains to evaluate the influence of transient intrauterine ischemia on the near-term foetus and to assess the effect of FK506 treatment.

Method Transient intrauterine ischemia was induced by 30 min of the right uterine artery occlusion at 17 days of gestation in Wistar rats. The vehicle or 1.0 mg·kg⁻¹ of FK506 was administered after 1 h of re-circulation. All of the pups were delivered by caesarean section at 21 days of gestation and samples of cerebral cortical tissue were obtained from pups at 1 h after birth. The mitochondrial respiration was measured polarographically in homogenates. For the analysis of ATP, ADP, and AMP, neonatal brains were frozen in situ and fluorometric enzymatic techniques were used.

Results In the neonatal cortical tissue exposed to ischemia, mitochondrial respiratory activities and ATP concentrations decreased significantly to about 59% and 67% of those in normoxic controls, respectively. The deterioration of both mitochondrial respiratory activities and high-energy phosphates was prevented by FK506, given 1 h after the start of re-circulation.

Conclusion The results indicate that the transient intrauterine ischemia is accompanied by mitochondrial dysfunction and

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